State of New Jersey

JON S. CORZINE
Governor

DEPARTMENT OF ENVIRONMENTAL PROTECTION
Trenton, NJ 08625-0409

LISA P. JACKSON
Commissioner

May 21, 2008

Water Docket
Environmental Protection Agency
Mail Code: 2822T
1200 Pennsylvania Ave, NW
Washington, DC 20460

RE: Docket ID No. EPA-HQ-OW-2007-1189
Drinking Water Contaminant Candidate List 3--Draft (Federal Register:February 21, 2008, Volume 73, Number 35, p. 9627-9654, DOCID:fr21fe08-134)

To Whom It May Concern:

In response to Federal Register notice of the February 21, 2008, the New Jersey Department of Environmental Protection (NJDEP) is pleased to comment on the Draft Contaminant Candidate List 3 (CCL3). Our comments focus on four chemicals of concern to New Jersey: PFOA, 1,2,3-trichloropropane, MTBE, and perchlorate.

General Comments

Section 1412(b) (1) of the federal Safe Drinking Water Act (SDWA), as amended in 1996, requires USEPA to publish the Contaminant Candidate List every five years. The SDWA specifies that the list must include contaminants that are not subject to any proposed or promulgated National Primary Drinking Water Regulations, are known or anticipated to occur in public water systems (PWSs), and may require regulation under SDWA. The 1996 SDWA Amendments also specify three criteria to determine whether a contaminant may require regulation: 1) the contaminant may have an adverse effect on the health of persons; 2) the contaminant is known to occur or there is a substantial likelihood that the contaminant will occur in public water systems with a frequency and at levels of public health concern; and 3) in the sole judgment of the Administrator, regulation of such contaminant presents a meaningful opportunity for health risk reduction for persons served by public water systems.

Since the Contaminant Candidate List process began about 10 years ago, none of the contaminants included on the two prior Contaminant Candidate Lists have been adopted by the United States Environmental Protection Agency (USEPA) as National Primary Drinking Water Regulations. The NJDEP is very concerned about this practice since many of the contaminants on the past Contaminant Candidate Lists, as well as many of the contaminants on the present CCL3, both occur widely in drinking water and have health effects of potential concern. The health effects data for many of these chemicals are extensive and complicated. For such chemicals, the risk assessment involves a large amount of time and effort. Most states have limited resources as far as risk

assessment and cannot address all chemicals of concern in drinking water without assistance from USEPA. We urge USEPA to use its vast resources to address such chemicals by regulating when appropriate, rather than simply determining that every chemical evaluated does not need regulation.

Specific Contaminants of Concern to New Jersey

Perfluorooctanoic acid (PFOA)

PFOA is a chemical of concern in drinking water, which NJDEP strongly urges USEPA to address, as discussed below. It is well known that drinking water has been contaminated by PFOA from point sources, such as the Ohio and West Virginia area contaminated by the DuPont Washington Works facility in Parkersburg, West Virginia and the several sites contaminated by 3M in Minnesota. However, it is also important to recognize that the lower levels of PFOA found in drinking water not impacted by a known point source may also result in health effects of concern.

As discussed below, it appears that health-based drinking water concentrations developed using a very conservative approach involving application of standard uncertainty factors for Reference Dose development, as well as animal-to-human extrapolation based on blood levels instead of administered dose, do not protect against health effects in the human population.

New Jersey Occurrence Information

In 2006, NJDEP conducted a small statewide PFOA and perfluorooctane sulfonic acid (PFOS) occurrence study (NJDEP, 2007a). Twenty-nine samples from twenty-three ground and surface water systems were collected in various areas throughout the state. STL Denver, the only laboratory certified by NJDEP for PFOA, conducted the analysis. The detection limit was 0.001 ug/L and the quantitation limit was 0.004 ug/L. Samples consisted of raw groundwater and surface waters, as well as samples of finished water. PFOA was found in raw and treated surface water, and raw and treated ground water from unconfined wells.

The data from the occurrence study show levels of PFOA from 0.003 ug/L, a level that is detected but not quantified to 0.039 ug/L. PFOA was detected and quantified at 65% of the 23 systems sampled. If the samples where PFOA was detected but was not quantifiable are included, PFOA was found in 78% of the systems sampled.

NJDEP is continuing to sample at water systems where PFOA was detected. In this follow-up sampling, PFOA was detected at four water systems at or above 0.04 ug/L (the NJDEP health-based drinking water guidance level, discussed below). The highest concentration was measured at systems with no known point source of PFOA was 0.069 ug/L.

Significance of Drinking Water Exposure to Human Body Burden

Although these drinking water concentrations of PFOA are relatively low, they may contribute significantly to the body burden of PFOA in the general population (Post et al., 2007). The average blood concentration of PFOA in the general population is approximately 5 ug/L and the 90th percentile is 9.4 ug/L (USEPA, 2005a).

A study of a community whose drinking water was contaminated with PFOA showed that, on average, ingestion of water with a given PFOA concentration results in a blood concentration 100-fold higher (Emmett et al., 2006). Similar results are predicted by modeling done by Gray (2005) and Hinderliter and Jepson (2001). For example, ingestion of drinking water with 0.03 ug/L PFOA

would be expected to contribute about 3 ug/L to the blood concentration of PFOA. Therefore, the contribution to total human exposure of PFOA in drinking water at levels below 0.1 ug/L may be significant.

Toxicology and Risk Assessment

New Jersey Health-based Drinking Water Guidance

In 2007, NJDEP developed a health-based drinking water guidance for PFOA of 0.04 ug/L in response to a request from a New Jersey water supply with detections of up to 0.19 ug/L in its wells, located near a point source of PFOA (NJDEP, 2007b).

The NJDEP health-based drinking water guidance is based on the endpoints identified by USEPA (2005a) in its draft PFOA risk assessment. Since the half life of PFOA is much longer in humans than in the experimental animals used in the studies, the risk assessment was based on blood levels of PFOA rather than administered doses of PFOA. This approach was recommended by the USEPA Science Advisory Board (USEPA, 2006a). Appropriate uncertainty factors and a Relative Source Contribution factor were applied to the blood levels in the animal studies at the NOAELs and LOAELs identified by USEPA to determine target human blood levels. A similar approach was used for the cancer endpoint to determine the blood level at the 10^{-6} risk level. The 100:1 blood/drinking water concentration factor observed by Emmett et al. (2006) was used to determine drinking water concentrations anticipated to result in the target blood levels.

The most sensitive endpoints, resulting in a health-based drinking water concentration of 0.04 ug/L, were body weight and hematological changes in a chronic study of adult female rats. The drinking water concentrations based on endpoints from several other studies were very close to 0.04 ug/L (adult male rat – 0.08 ug/L, cynomolgus monkey – 0.05 ug/L, pregnant female rat – 0.07 ug/L, carcinogenicity in male rats at 10^{-6} risk level – 0.06 ug/L).

The USEPA (2005a) draft risk assessment did not consider more recent studies, which reveal additional effects of PFOA of serious concern, as discussed below.

Recent mouse developmental data

The developmental data considered by USEPA (2005a) were from rat studies. The rat is not a good model for developmental effects of PFOA because the half-life of PFOA in female rats is very short (2-4 hours), so that steady state is never reached with daily dosing, and the fetus receives minimal exposure. In contrast, PFOA has a longer half-life in female mice, resulting in a higher steady state blood level and greater exposure to the fetus. Recent mouse developmental studies, reviewed by Lau et al. (2007), have revealed effects resulting from prenatal exposure including post-natal mortality, decreased birth weight, delayed post-natal growth and development, obesity at 18 months of age, and inhibition of mammary gland development.

Recent data on mechanism of liver carcinogenicity

Very recent new data shed light on the relevance of the positive carcinogenicity studies in rats to PFOA's human carcinogenic potential, which has been a subject of ongoing controversy.

PFOA was found to induce liver adenomas, Leydig cell adenomas, and pancreatic acinar cell tumors in male Sprague-Dawley rats and mammary cell fibroadenomas in female rats, although USEPA (2005a) questioned the significance of the mammary tumors compared to historical background

incidences. USEPA (2005a) interpreted the liver, Leydig cell, and pancreatic acinar cell tumors as constituting a "tumor triad" known to be caused by a class of chemicals which are peroxisome proliferator-activated receptor-alpha (PPAR-α) agonists. USEPA (2005a) states that the relevance to humans of tumors caused by this group of chemicals is uncertain. Additionally, they state that there are no adequate human studies of PFOA's carcinogenic potential since the occupationally exposed cohorts are still quite young. Based upon this, USEPA (2005a) concluded that PFOA was best described as having "suggestive evidence of carcinogenicity, but not sufficient to assess human carcinogenic potential".

The USEPA Science Advisory Board (2006a) disagreed with USEPA (2005a) as to the most appropriate descriptor for the carcinogenicity of PFOA. They concluded that the PFOA cancer data are consistent with the USEPA cancer guidelines descriptor "likely to be carcinogenic to humans" which applies to chemicals which have been shown to cause tumors in more than one species, sex, strain, site, or exposure route, regardless of the existence of evidence in humans. Most Science Advisory Board panel members felt that the animal data are much stronger than the type of data which would warrant a "suggestive" descriptor. This conclusion was based on the following: 1) existence of two positive cancer studies in animals for PFOA, 2) data suggesting that PPAR-α activation may not be the sole mechanism of liver carcinogenesis (discussed above in regard to liver toxicity of PFOA), 3) disagreement about the appropriateness of comparison to historical controls rather than concurrent controls in evaluating the mammary tumor date, and 4) disagreement about the USEPA (2005a) assumptions about the mode of action for the Leydig cell and pancreatic acinar cell being related to PPAR-alpha agonist activity, and the USEPA comparison of mammary tumors to historical, rather than concurrent, controls.

A new study (Tilton et al., 2008) suggests that PFOA promotes liver tumors in rainbow trout by a mechanism independent of PPAR-alpha and peroxisome proliferation. The rainbow trout has been used as a model for chemically induced liver cancer in humans for over 40 years, and, like humans, is insensitive to liver toxicity through peroxisome proliferation. PFOA was shown to enhance liver carcinogenesis post-initiation through an estrogenic mechanism independent of peroxisome proliferation, which may be relevant to human carcinogenic potential.

Recent human birth weight and clinical effects at very low exposures
Recent human data suggest effects in humans at blood levels of PFOA typical of the general population, orders of magnitude below effect levels seen in animal studies.

Several studies associating PFOA blood levels in the general population with effects on birth weight and other fetal growth indicators (ponderal index, head circumference, and birth length) have been published (Apelberg et al., 2007; Fei et al. 2007; Fei et al., 2008). The effects observed within the general population are of a very large magnitude. For example, in a study of 1400 Danish mothers and their infants (Fei et al., 2007), the largest decrease in mean birth weight, 126 g, was seen between the lowest quartile for PFOA in maternal blood (3.91 ug/L or below) and the second quartile (3.91-5.20 ug/L). The decrease in mean birth weight between the lowest quartile (less than 3.91 ug/L and the highest quartile (6.97 ug/L and above) was 175 g. No association between birth weight and PFOS blood levels was observed.

Additionally, recent preliminary data from a study of approximately 70,000 Ohio and West Virginia residents exposed to PFOA in their drinking water at levels from 0.05 ug/L to 3 ug/L or higher suggest an association between several clinical parameters and exposure to PFOA. Apparent effects

are seen at blood levels orders of magnitude below the blood levels at which effects have been seen in animals (Frisbee, 2008). The effects involved include decreased C-reactive protein (an indicator of inflammation), decreased immunoglobulin G, increases in two liver enzymes (SGPT and SGOT), increased cholesterol in children, and effects on thyroxine levels. These effects are consistent with the known toxicity of PFOA from animal and/or human occupational studies, as reviewed by Lau et al. (2007), as PFOA is known to produce liver toxicity, suppress the immune system, and suppress inflammatory response in animal studies, and has been observed to increase thyroid hormone and cholesterol levels in occupational studies.

In this study, the study population was divided into deciles based on PFOA blood levels, with about 6700 people in each group. The mean blood levels for each decile is shown below (S. Frisbee, personal communication):

Decile	**Mean PFOA Blood Level (ug/L)**
1	5.65
2	9.80
3	13.47
4	18.06
5	24.28
6	33.26
7	47.44
8	71.33
9	124.11
10	482.33

The PFOA levels in the first decile and the second decile are almost identical to the mean and the 90th percentiles in the United States population. No threshold was apparent for a number of the observed effects in human populations, both for the birth weight study (Fei et al., 2007) and for clinical parameters (C-8 reactive protein, liver enzymes, and increased cholesterol in children), and effects appear to occur down to the lowest exposure groups, which are in the range of the mean concentration general population.

Effects are seen well below the blood levels derived by applying conservative uncertainty factors to animal data by NJDEP (2007b); the blood level for the most sensitive effect thus derived was 18 ug/L.

Recommendation

As discussed above, it appears that health-based drinking water concentration for PFOA developed using a very conservative approach involving application of standard uncertainty factors for Reference Dose development, as well as animal-to-human extrapolation based on blood levels instead of administered dose, is not protective against health effects in the human population. Because humans appear to be so sensitive to PFOA's effects and PFOA appears to occur ubiquitously in drinking water, based on New Jersey and other data, it is important that exposure to PFOA through drinking water be appropriately limited through MCL development.

1,2,3-Trichloropropane

1,2,3-Trichloropropane (1,2,3-TCP) is a chemical of concern in drinking water, which NJDEP strongly urges USEPA to address. 1,2,3-TCP is a potent genotoxic carcinogen which occurs in drinking water at levels resulting in significant cancer risk based on test results from New Jersey public and non-public water supplies. Currently, USEPA does not provide any information as to 1,2,3-TCP's carcinogenicity on its 2006 USEPA Office of Water Drinking Water Regulations and Health Advisory Table (USEPA, 2006b). Instead, it provides a Lifetime Health Advisory which results in a lifetime cancer risk of 4×10^{-3} to 3×10^{-2}, based on available slope factors. This information, which is not protective of public health, has been used by several states to develop drinking water and groundwater guidance for 1,2,3-TCP, as the basis for evaluating situations of drinking water and groundwater contamination, etc. Therefore, NJDEP strongly urges USEPA to provide appropriate public health protective information in its Drinking Water Regulations and Health Advisory Table and to develop an MCL for 1,23-trichloropropane.

Uses

1,2,3-TCP is a known contaminant of nematocides and soil fumigants including D-D (NTP, 2005) (1,2-dichloropropane and 1,3-dichloropropene [mixed isomers]) and Telone (1,3-dichloropropene). Telone has been reported to contain up to 0.17% 1,2,3-TCP by weight (WHO, 2003). This is thought to be the source of 1,2,3-TCP contamination of rural wells in New Jersey and other locations. When 1,2,3-TCP was detected in Galloway Township, Atlantic County, New Jersey, in 1999, three other chemicals were also found at the sampling sites: dibromochloropropane, 1,2-dichloropropane, and ethylene dibromide. The occurrence of 1,2,3-TCP is discussed in more detail.

1,2,3-TCP was extensively used in the past as a solvent, cleaning and degreasing agent, and paint and varnish remover. It is used as intermediate in the synthesis of several organic compounds including polysulfone liquid polymers, dichloropropene, hexafluorpropylene, and polysulfides (NTP, 2005).

1,2,3-TCP is a byproduct produced in significant quantities in the manufacture of other chlorinated compounds, including epichlorohydrin. It is listed as a component which is present at greater than 0.01% (a reporting threshold) on the Right to Know lists of New Jersey, Pennsylvania, and Massachusettes, as well as the California Proposition 65 list. According to the SPI Epichlorohydrin Task Force, the majority of 1,2,3-TCP produced as a byproduct of epichlorohydrin production today is incinerated on-site (WHO, 2003). 1,2,3-TCP is also a byproduct of dichloropropene, propylene dichlorohydrin, dichlorohydrin, and glycerol (NTP, 2005).

A polymer used as a coagulant in the treatment of potable water and wastewater is produced by reacting epichlorohydrin with dimethylamine. Epichlorohydrin is a known contaminant of these polymers, and is likely to be present in finished drinking water due to its use in the coatings of drinking water pipes (USEPA, 1985). Since 1,2,3-trichloropropane is a contaminant in epichlorohydrin, it might also be present in drinking water, especially since 1,2,3-trichloropropane is more stable in water than epichlorohydrin.

An NSF International (2000) report prepared for Health Canada on impurities in drinking water treatment, 1,2,3-TCP was identified as a contaminant in an unidentified well drilling aid. However, the Action Level (target drinking water level based on health effects) used by Health Canada was 5 ug/L, which is much higher than the health based drinking water level or practical quantitation limit (see below).

A health-based drinking water guidance of 0.005 ug/L was developed by NJDEP in 1999 based upon the 10^{-6} risk level and the slope factor of 7 $(mg/kg/day)^{-1}$ in the USEPA HEAST (1995) tables. This slope factor was based on the combined incidence of benign and malignant tumors at various sites in rats. Standard exposure factors for body weight (70 kg) and drinking water ingestion (2 L/day) were used. The New Jersey Drinking Water Quality Institute is currently in the process of recommending a Maximum Contaminant Level (MCL) to NJDEP for 1,2,3-TCP for possible standard setting. A more complete explanation of the risk assessment is presented below.

Occurrence

1,2,3-Trichloropropane has been found in both public water supplies (including in USEPA Unregulated Contaminant Monitoring) and private wells. As discussed above, it is known to be present in soil fumigants including D-D (mixed isomers of 1,2-dichloropropane and 1,3-dichloropropene) and Telone (1,3-dichloropropene), and there are also other potential sources of contamination. Because the health-based drinking water concentration (at the10^{-6} risk level) for 1,2,3-trichloropropane is so low (see below), less sensitive analytical methods will miss occurrences at levels of concern, and reporting limits given by each state for the UCMR Round 1 monitoring ranged from 0.5 ug/L to 5 ug/L.

In 2005, NJDEP conducted a review of occurrence of 1,2,3-trichloropropane in New Jersey drinking waters. 1,2,3-Trichloropropane was detected at levels above the health-based drinking water guidance developed by NJDEP of 0.005 ug/L (see below) in 30 of the 2,640 private wells (1.1%) sampled during contaminated site investigations overseen by NJDEP's Site Remediation Program between 1999 and 2004. In addition, as part of NJDEP's Synthetic Organic Compound (SOC) Waiver Program sampling, 1,2,3-trichloropropane was detected at levels above the health-based drinking water guidance of 0.005 ug/L developed by NJDEP in 11 of approximately 260 community water systems (4%) sampled between 1999 and 2004.

In unregulated contaminant monitoring in California from 1989 through the 1990s, fewer than 20 sources reported detections, reflecting the less sensitive analytical methods available at that time, with a reporting limit of 0.5 µg/L. However, more recent monitoring in California with a more sensitive analytical method reported 1,2,3-trichloropropane detections in 303 sources, at levels up to 57 µg/L. Of the 303 detections, 2 were below 0.005 ug/L, 171 between 0.005 and 0.05 ug/L, 104 between 0.05 and 0.5 ug/L, 20 between 0.5 and 5 ug/L, 4 between 5 and 50 ug/L, and 1 above 50 ug/L. This dataset is not yet complete (CDPH, 2007).

California Department of Public Health (CDPH, 2007) states on its fact sheet that the primary possible contaminating activity for 1,2,3-TCP in drinking water appears to be hazardous waste sites. No further justification is given for this statement.

1,2,3-Trichloropropane is the main contaminant at a Superfund site, MacKenzie Chemical Works in Suffolk County, New York (ATSDR, 2004). ATSDR conducted an evaluation of this site in 2004, and used New York's MCL of 5 ug/L to evaluate the significance of the contamination. (5 ug/L is New York's MCL for Principal Organic Contaminants, a generic number for organic contaminants when there is no information indicating that a lower value is warranted.) The cancer risk level at 5 ug/L is about 10^{-3}. Analytical methods used in this investigation were chosen with the belief that a detection limit below 5 ug/L was not needed.

According to the ATSDR report on this site, concentrations of 1,2,3-trichloropropane of up to 34,000 ug/L were detected in off-site groundwater, at 10,000 ug/L one block from the site, and above 100 ug/L more than 0.25 miles from the site. A well field is located about 0.5 miles downgradient from the site. No VOCs were detected in routine monitoring of a deep well in that well field that serves the local community. ATSDR concluded that "the site poses no public health hazard at the present because there are no known exposures to site-related contaminants." It is troubling that a drinking water benchmark of 5 ug/L, which is 1000 times above the public health protective concentration of 0.005 ug/L, was used by ATSDR and the state of New York to evaluate this highly contaminated site. This information was used to provide reassuring, but perhaps incorrect, conclusions to the local community that there is no exposure of concern to 1,2,3-trichloropropane.

Additional test results posted on web sites reveal many other cases of drinking water and ground water contamination by 1,2,3-trichloropropane at high levels, including a site in Dover Township, New Jersey where concentrations up to 210 ug/L in ground water were found (NJDHSS, 2001).

Toxicology and Risk Assessment

1,2,3-Trichloropropane is metabolically activated to a reactive intermediate and is a potent carcinogen. It is genotoxic in almost every system in which it was tested, and it caused a high incidence of tumors at multiple sites in male and female rats and mice in an NTP (1993) carcinogenicity bioassay. From the 1993 study, NTP concluded that there was clear evidence of carcinogenic activity in both sexes of both species. Additionally, it was reasonably anticipated to be a human carcinogen in the NTP Eleventh Report on Carcinogens (2005). Thus, the weight of evidence for carcinogenicity for 1,2,3-trichloropropane is much stronger than for many other chemicals which are treated as non-threshold carcinogens as a conservative default assumption. From this information, there is no doubt that the risk assessment for 1,2,3-trichloropropane should be based on a non-threshold low dose model, and that a risk assessment based on a Reference Dose is inappropriate for this chemical.

Three slope factors have been developed for 1,2,3-trichloropropane using the data from the 1993 NTP study: an older USEPA HEAST (1995) slope factor which was based on classification as a probable human carcinogen (B2) and the linearized multistage model, and slope factors in the draft risk assessments developed by USEPA IRIS (2007) and the California EPA Public Health Goal program (2007) which both use time-to-tumor low dose modeling, but differ in choice of species and modeling details. The range of drinking water concentrations at the 10^{-6} risk level, based on the three slope factors available, is 0.0015 ug/L to 0.009 ug/L, assuming water consumption of 2 liters per day.

A health-based drinking water guidance of 0.005 ug/L was developed by NJDEP in 1999 based upon the 10^{-6} risk level and the slope factor of 7 $(mg/kg/day)^{-1}$ in the USEPA HEAST (1995) tables. This slope factor was based on the combined incidence of benign and malignant tumors at various sites in rats. Standard exposure factors for body weight (70 kg) and drinking water ingestion (2 L/day) were used. The New Jersey Drinking Water Quality Institute is currently in the process of developing a Maximum Contaminant Level recommendation to NJDEP for 1,2,3-TCP.

Health Advisory information provided by USEPA Office of Water

The 2006 USEPA Office of Water Drinking Water Regulations and Health Advisory Table (HA Table) does not indicate that 1,2,3-trichloropropane is a carcinogen and provides a Lifetime Health Advisory for this contaminant. As you know, the users of this table do not necessarily have any

toxicology or risk assessment expertise, since it is a practical reference table intended to be used by persons directly addressing local public health issues, such as detection of drinking water contaminants without MCLs in public water supplies or private wells. Examples of users of the HA Table are county and local health officials or water purveyors.

As discussed above, there is no doubt that the cancer risk assessment for 1,2,3-trichloropropane should be based on a non-threshold linear low dose model, and that a risk assessment based on a Reference Dose is inappropriate for this chemical. In spite of this, the IRIS entry for 1,2,3-TCP, **fifteen years** after the 1993 NTP study was published in final form, provides a Reference Dose for non-cancer effects of 0.006 mg/kg/day which was developed in 1989, prior to completion of the 1993 NTP study. For carcinogenicity, the only entry is "no data, dated 11/1/1993."

USEPA Office of Water does not provide Lifetime Health Advisories for known or probable human carcinogens (Groups A or B, using the older weight of evidence terminology). However, the HA Table does not even indicate that 1,2,3-trichloropropane is a carcinogen, and gives a Lifetime Health Advisory of 40 ug/L, based on the IRIS Reference Dose of 0.006 mg/kg/day. The range of lifetime cancer risks at the Lifetime Health Advisory of 40 ug/L, using the slope factors currently being considered by USEPA and California EPA is 4×10^{-3} to 3×10^{-2} or **4 in 1000** to **3 in 100**. Thus, an unacceptably high level of cancer risk will result if the guidance provided in the table is used by local health officials to provide advice about 1,2,3-trichloropropane contamination of drinking water. Additionally, this guidance may be used as the basis to choose analytical methods which are not sensitive enough to detect levels of concern.

Current USEPA Office of Water procedures do not allow information from final NTP studies, HEAST, NCEA, IRIS drafts, or other such reliable sources, to be posted on the HA Table, even if this information clearly demonstrates that the information provided on the HA Table is not protective of public health. USEPA Office of Water procedures do not allow for inclusion of any carcinogenicity information until the IRIS cancer assessment for this chemical is finalized, which will likely take years due to the number of time consuming reviews required by the IRIS process.

Ironically, older versions of the Drinking Water Regulations and Health Advisory Table (e.g. 1996) listed 1,2,3-trichloropropane as a Group B2 (probable human) carcinogen and provided a drinking water concentrations based on cancer risk, based on the slope factor provided in the 1995 HEAST table. As discussed above, the HEAST slope factor was the basis for the drinking water guidance value of 0.005 ug/L developed by NJDEP in 1999.

The Lifetime Health Advisory of 40 ug/L provided in the HA Table is widely used by states and other parts of USEPA as a health-based drinking water concentrations for 1,2,3-trichloropropane. Some examples follow:

1) Situations where Lifetime Health Advisory from the HA Table has been routinely used by state and local environmental or public health officials in states with no listed guidance to assess the significance of detections in drinking water. It is obviously not possible to determine how many times that this has occurred, since this information is not normally posted on the Internet.

2) Several states, including Arizona (42 ug/L), Florida (42 ug/L), Minnesota (40 ug/L), New York (5 ug/L) Washington (21 ug/L), and Wisconsin (50 ug/L) have drinking water standards

or guidance concentrations at high risk levels, based on the USEPA's incorrect and outdated toxicity information (USEPA, 2000; HSDB, 2008).

3) Because the health-based drinking water concentration (at the 10^{-6} risk level) for 1,2,3-trichloropropane is so low, less sensitive analytical methods will miss occurrences at levels of concern, and reporting limits given by each state for the Unregulated Contaminant Monitoring Round 1 monitoring ranged from 0.5 ug/L to 5 ug/L.
Additionally, the 2001 USEPA Office of Water reports "Occurrence of Unregulated Contaminants in Public Water Systems-A National Summary" (Table V.A.1) (USEPA, 2001a) and "Occurrence of Unregulated Contaminants in Public Water Systems-An Initial Assessment" (Tables A58 a, c) (USEPA, 2001b) incorrectly give 40 ug/L as the "MCL" for 1,2,3-trichloropropane, although it is currently not a regulated contaminant.

Recommendations
NJDEP strongly urges USEPA Office of Water to update its HA Table to provide appropriate public health protective drinking water guidance based on the carcinogenic effects of 1,2,3-TCP. NJDEP strongly urges USEPA Office of Water to develop an MCL for 1,2,3-TCP based on its occurrence in drinking water and its carcinogenic effects.

Methyl tertiary butyl ether (MTBE)
MTBE is a chemical of concern in drinking water, which NJDEP strongly urges USEPA to address, as discussed below. MTBE was listed on CCL1 and CCL2, and both times a decision was made not to regulate this chemical. In addition, MTBE is included in the current CCL3. There is as much or more relevant toxicology data on MTBE than for many other regulated chemicals, and it is a contaminant which is commonly found in drinking water throughout the United States. At least 41 of 50 states have developed a drinking water or ground water standard or guidance for MTBE as shown on a map linked from the USEPA website (http://www.epa.gov/swerust1/mtbe/mtbemap.pdf). MTBE is the most frequently detected volatile organic contaminant in data collected through New Jersey's Private Well Testing Act. Of the 51,028 wells tested under the Private Well Testing Act, 38 (0.01%) exceeded the New Jersey MCL of 70 ug/L and the highest concentration detected was 1550 ug/L.

New Jersey was among the first states to develop an MCL for MTBE. The basis for New Jersey's MCL was published in 1994 (NJDWQI, 1994), and it was adopted in 1996.

There is controversy over the reporting and interpretation of the results of the only available chronic oral study for MTBE (Belpoggi et al.,1995; Belpoggi et al., 1998). In this study, rats were dosed with MTBE by gavage in olive oil. The New Jersey Drinking Water Quality Institute (1994) recognized the uncertainties regarding the carcinogenicity of MTBE, specifically by oral exposure, and classified MTBE as a possible human carcinogen (equivalent to USEPA Group C under the 1986 USEPA guidelines). This classification forms the basis for New Jersey's Drinking Water Health-based Maximum Contaminant Level (MCL) for MTBE. The Health-based MCL is based on a subchronic oral gavage study in rats (Robinson, 1990), and the endpoint of concern was increased relative kidney weight. An additional uncertainty factor of 10 was incorporated to account for possible carcinogenic effects, following USEPA's and New Jersey's approach for drinking water risk assessment of possible human carcinogens. Some states have followed New Jersey's approach, while other states have based their MTBE risk assessments on a cancer slope factor.

A subchronic drinking water study for MTBE in rats was recently conducted (Bermudez et al., 2008) in order to set the doses for a two year drinking water study. This two year study is currently in progress, with the in-life phase expected to end in April 2009. The target date for the final report for this study is January 2010 (E. Bermudez, personal communication). The results of this study may answer some of the unresolved issues regarding the oral carcinogenicity of MTBE.

Recommendation

MTBE is a contaminant for which the states have a strong need for guidance from USEPA. It is recommended that USEPA evaluate the results of the current chronic oral drinking water study when it is complete, along with other toxicology information, and develop an MCL for MTBE.

Perchlorate

Perchlorate is a chemical of concern in drinking water, which NJDEP strongly urges USEPA to address, as discussed below. Perchlorate is a chemical with diverse uses in flares, fireworks, rocket fuel, and explosives. It is also found in fertilizers from certain geographic regions. Its widespread occurrence in drinking water has been well documented by others. After perchlorate was found in New Jersey drinking water as part of monitoring required by USEPA's Unregulated Contaminant Monitoring Rule, the New Jersey Drinking Water Quality Institute was asked by NJDEP to recommend a drinking water standard for perchlorate. NJDEP anticipates proposing a perchlorate MCL of 5 ug/L in the near future, as discussed below.

The National Research Council (2004) recommended a Reference Dose for perchlorate based on a small, but detailed study by Greer et al. (2002), which measured the inhibition of iodine uptake by the thyroid and the levels of thyroid hormone in adults receiving doses of perchlorate. The No Observed Effect Level for inhibition of iodine uptake in this study was 0.007 mg/kg/day. Iodine uptake is a full biological step away from a reduction in thyroxine or an increase in TSH, neither of which was observed even at the highest dose of perchlorate in this study.

The Reference Dose of 0.0007 mg/kg/day incorporates an uncertainty factor of 10 to protect sensitive subpopulations including fetuses and infants. Additional uncertainty factors were not considered necessary since the observed effect was not in and of itself considered adverse. This conclusion was supported by other data indicating the absence of effects on thyroid hormone in newborns and pregnant women in areas with perchlorate in the drinking water. Additional data since then has confirmed this for both groups. This Reference Dose was adopted by USEPA IRIS in 2005.

After reviewing the scientific literature the New Jersey Drinking Water Quality Institute concurred with National Research Council's Reference Dose (NJDWQI, 2005). From this Reference Dose, the Institute established a drinking water equivalent level of 24.5 micrograms of perchlorate per liter of water based on a 70 kilogram adult.

The last step in developing a health-based drinking water concentration is to adjust the drinking water equivalent level to account for other sources of exposure, such as food. When little data is available on other exposures that may add to the exposure from water, the default assumption is that the drinking water contribution should be limited to 20% of the total. Application of a 20% Relative Source Contribution factor results in a health-based drinking water concentration of 5 ug/L, which was recommended as the basis for New Jersey's MCL (NJDWQI, 2005). Based on their relative intakes of water and formula or breast milk, infants were considered to be protected. This is

supported by the recent publication of Food and Drug Administration data on exposure to perchlorate in foods.

Recommendation
NJDEP strongly urges USEPA to develop an MCL for perchlorate based on its widespread occurrence, well characterized effects on thyroid function, and adequate information on the relative contributions of food and drinking water to perchlorate exposure.

Other CCL3 Contaminants Addressed by NJDEP
New Jersey has developed Health-based MCLs (equivalent to USEPA MCLGs) for formaldehyde, n-hexane, and ethylene glycol, and is currently in the process of developing analytical methods for these so that an MCL may be adopted. The basis for the formaldehyde and n-hexane Health-based MCLs is found on the New Jersey Drinking Water Quality Institute's website http://www.nj.gov/dep/watersupply/njdwqinstitute.htm. A recommendation has been made to revise the Health-based MCL for ethylene glycol from 290 ug/L to 10,000 ug/L, based on evaluation of more recent data on both renal toxicity and developmental effects.

New Jersey promulgated an MCL for 1,1-dichloroethane in 1996. The New Jersey carcinogenicity classification, Reference Dose, and Health-based MCL for 1,1-dichloroethane, which were developed in 1994 and are found on the website given above, were recently reevaluated by the New Jersey Drinking Water Quality Institute. Based on reevaluation of the data relevant to carcinogenic effects and the current cancer risk assessment guidelines, it is recommended to classify 1,1-dichloroethane in New Jersey Carcinogenicity Category II, equivalent to Group C, under the previous (1986) USEPA guidance, and analogous to Suggestive Evidence of Carcinogenic Potential under the current (2005) guidance. USEPA IRIS does not provide a slope factor for 1,1-dichloroethane. It was recommended that an additional uncertainty factor of 10 for potential carcinogenicity be incorporated into the Reference Dose.

The current basis of the Reference Dose, kidney effects in cats exposed by inhalation, is a more sensitive endpoint than a newer rat oral subchronic study, and it was recommended to continue to use the current endpoint. It is recommended that the uncertainty factor of 5 currently used for small number of animals in the cat study be removed. The basis for the Reference Dose is already very conservative and health protective and the total uncertainty factor would exceed the maximum uncertainty factor of 10,000 used in Reference Dose development.

Based on the above, the recommended Reference Dose is 0.0032 mg/kg/day, which is a two-fold decrease from current Reference Dose of 0.0065 mg/kg/day. The recommended Health-based MCL is 23 ug/L, also a two-fold decrease from the current value of 46 ug/L.

Conclusion
We appreciate the opportunity to comment on the Draft Contaminant Candidate List 3. If you have any questions or require additional information, please contact Dr.Gloria Post of the NJDEP Division of Science, Research and Technology at (609) 292-8497 or gloria.post@dep.state.nj.us.

Attachment

Sincerely,

Judith Louis, Ph.D., Chief
Bureau of Environmental Assessment
Division of Science Research and Technology

Barker Hamill, Assistant Director
Water Supply Operations
Division of Water Supply

C: Eileen Murphy, NJDEP
Michele Putnam, NJDEP
Sandra Krietzman, NJDEP
Gloria Post, NJDEP
Mark Robson, Chair, New Jersey Drinking Water Quality Institute

Attachment

Citations

Appelberg, B.J., Witter, F.R., Herbstman, J.B., Calafat, A.M, Halden, R.U., Needham, L.L, and Goldman, L.R. (2007). Cord serum concentrations of perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA) in relation to weight and size at birth. Environmental Health Perspectives 115 (11), 1670-1676.

ATSDR (2004). Agency for Toxic Substances & Disease Registry. Public Health Assessment. Former Mackenzie Chemical Works Site. Central Islip, Suffolk County, New York. EPA Facility ID: NYD980753420. September 29, 2004. http://www.atsdr.cdc.gov/HAC/pha/mackenzie092904/mackenzie092904-toc.html

Belpoggi, F., Soffaritti, M., and Maltoni, C. (1995). Methyl tertiary-butyl ether (MtBE) - a gasoline additive - causes testicular and lymphohaematopoietic cancers in rats. Toxicology and Industrial Health 11 (2): 119-149.

Belpoggi, F., Soffritti, M., and Maltoni, C. (1998). Pathological characterization of testicular tumours and lymphomas-leukemias, and of their precursors observed in Sprague-Dawley rats exposed to methyl-tertiary-butyl-ether (MTBE). Eur. J. Oncol. 3 (3): 201-206.

Bermudez, E., Parkinson, H.D., and Dodd, D.E. (2008). Methyl tertiary butyl ether (MTBE) 13-week drinking water study in Wistar rats. The Toxicologist, Abstract 508, p.104.

California Department of Public Health (2007). 1,2,3,-Trichloropropane. Last update: December 26, 2007. http://www.cdph.ca.gov/certlic/drinkingwater/Pages/123TCP.aspx

California Environmental Protection Agency(2007). Draft Public Health Goal for 1,2,3-Trichloropropane in Drinking Water. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. September 2007. http://www.oehha.org/water/phg/pdf/TCPphg091407.pdf

Emmett, E.A., Shofer, F.S., Zhang, H., Freeman, D., Desai, C., and Shaw, L.M. (2006). Community exposure to perfluorooctanoate: Relationships between serum concentrations and exposure sources. Journal of Occupational and Environmental Medicine 48 (8): 759-770.

Fei, C., McLaughlin, J.K., Tarone, R.E., and Olsen, J. (2007). Perfluorinated chemicals and fetal growth: A study within the Danish National Birth Cohort. Environmental Health Perspectives 115 (11), 1677-1682.

Fei, C., McLaughlin, J.K., Tarone, R.E., and Olsen, J. (2008). Fetal growth indicators and perfluorinated chemicals: A study in the Danish National Birth Cohort. Environmental Health Perspectives. American Journal of Epidemiology. doi:10.1093/aje/kwn095, May 5, 2008 Advance Access.

Frisbee, S. (2008). The C8 Health Project: How a class action lawsuit can interact with public health – history of events. West Virginia University School of Medicine Department of Community Medicine Public Health Grand Rounds. May 7, 2008. http://www.hsc.wvu.edu/som/cmed/ophp/pdfs/Public%20Health%20Grand%20Rounds%2005-07-2008%20-%20C8%20Health%20Project.pdf

FSTRAC (2000). Federal-State Toxicology and Risk Analysis Committee. Summary of state and federal drinking water standards and guidelines, 1998-1999. Cosponsored by Office of Science and Technology, Office of Water, United States Environmental Protection Agency.

Gray, D. (2005). Comments on the draft revision of Minnesota's Health Risk Limits (HRLs) for groundwater. Letter from Tetra Tech Inc. to Minnesota Department of Health. November 3, 2005.

Greer, M.A., Goodman, G., Pleus, R.C., and Greer, S.E. (2002). Health effects assessment for environmental perchlorate contamination: the dose response for inhibition of thyroidal radioiodine uptake in humans. Environmental Health Perspectives 110, 927-937.

HSDB (2008). Hazardous Substances Data Bank. 1,2,3-Trichloropropane. CASRN: 96-18-4. May 19, 2008.

Hinderliter, P.M. and Jepson, G.W. (2001). A simple conservative compartmental model to relate ammonium perfluorooctanoate (APFO) exposure to estimate of perfluorooctanote (PFO) blood levels in humans (draft). DuPont Haskell Laboratory for Health and Environmental Sciences. October 10, 2001.

Lau, C., Anitole, K., Hodes, C., Lai, D., Pfahles-Hutchens, A., and See, J. (2007). Perfluoroalkyl acids: a review of monitoring a toxicological findings. Toxicological Science 99 (2), 366-394.

NSF International (2000). Review of Contaminant Occurrences in Drinking Water Treatment Chemicals. A Summary Report of NSF International Results (1991 to 1999) ANSI/NSF Standard 60: Drinking Water Treatment Chemicals– Health Effects. Report Date: March 31, 2000. Prepared for: Health Canada,Health Protection Branch. http://www.hc-sc.gc.ca/ewh-semt/water-eau/drink-potab/mater/nsf_chemicals-chimiques_e.html

NRC (2005). National Research Council. Health Implications of Perchlorate Ingestion. Board on Environmental Studies and Toxicology, Division on Earth and Life Sciences, National Research Council, Washington, DC.

NTP (1993). Toxicology and carcinogenesis of 1,2,3-trichloropropane (CAS No. 96-18-4) in F344/N Rats and B6C3F1 Mice (gavage studies). Technical Report Series No 384. NIH publication No. 94-2839. Research Triangle Park, NC: National Toxicology Program. 348 pp.

NTP (2005). 1,2,3-Trichloropropane, Report on Carcinogens, Eleventh Ed.; U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program, January 2005.

NJDEP (2007a). New Jersey Department of Environmental Protection. Determination of perfluorooctanoic acid (PFOA) in aqueous samples. Final report. http://www.nj.gov/dep/watersupply/pfoa.htm

NJDEP (2007b). New Jersey Department of Environmental Protection. Guidance for PFOA in drinking water at Pennsgrove Water Supply Company. Memorandum from Gloria Post to Barker Hamill. http://www.nj.gov/dep/watersupply/pfoa.htm

NJDHSS (2001). New Jersey Department of Health and Senior Services. Public Health Assessment. Ciba-Geigy Corporation. CERCLIS Number: NJD001502517. Dover Township, Ocean County, New Jersey. March 12, 2001. http://www.state.nj.us/health/eoh/assess/cgc_pha_fnl.pdf

NJDWQI (1994). New Jersey Drinking Water Quality Institute. Maximum Contaminant Level. Recommendations for Hazardous Contaminants in Drinking Water. Appendix A: Health-based Maximum Contaminant Level Support Documents. September 26, 1994. Methyl tertiary butyl ether. Page A-31. http://www.nj.gov/dep/watersupply/njdwqinstitute.htm

NJDWQI (2005). New Jersey Drinking Water Quality Institute. Maximum Contaminant Level Recommendation for Perchlorate. Submitted to New Jersey Department of Environmental Protection. October 7, 2005. http://www.nj.gov/dep/watersupply/njdwqinstitute.htm

Post, G., Boros-Russo, B., and Fell, K. (2007). PFOA in public water supplies may contribute to the exposure of the general population. Presented at: Society of Toxicology Contemporary Concepts in Toxicology. Perfluoroalkyl Acids and Related Chemistries. Toxicokinetics and Mode of Action. February 14-16, 2007. Arlington, VA.

Robinson, M., Bruner, R.H., and Olson, G.R. (1990). Fourteen and ninety day toxicity studies of methyl tertiary-butyl ether in Sprague-Dawley rats. Journal of the American College of Toxicology 9 (5): 525-540.

Tilton, S.C., Orner, G.A., Benninghoff, A.D., Carpenter, H.M., Hendricks, J.D., Pereira, C.B., and Williams, D.E. (2008). Genomic Profiling Reveals an Alternate Mechanism for Hepatic Tumor Promotion by Perfluorooctanoic Acid in Rainbow Trout. Environmental Health Perspectives. doi:10.1289/ehp.11190. (May 9, 2008 In-Press).

USEPA (1985). United States Environmental Protection Agency. National Primary Drinking Water Regulations; Synthetic Organic Chemicals, Inorganic Chemicals and Microorganisms; Proposed Rule. Fed. Reg. 50 (219). Pages 46936-47022. November 13, 1985.

USEPA (1986). United States Environmental Protection Agency. Guidelines for Carcinogen Risk Assessment. Federal Register 51(185):33992-34003. September 24, 1986. http://www.epa.gov/ncea/raf/car2sab/guidelines_1986.pdf

USEPA (1995). United States Environmental Protection Agency. Health Effects Advisory Summary Tables (HEAST). FY 1995 Update. Office of Solid Waste and Emergency Response, 9200.6-303 (95-1), EPA-540-R-95-036. May 1995.

USEPA (1996). United States Environmental Protection Agency. 1996 Edition of the Drinking Water Standards and Health Advisories. Office of Water. October 1996.

USEPA (2001a). United States Environmental Protection Agency. Occurrence of Unregulated Contaminants in Public Water Systems-A National Summary. Office of Water. EPA 815-P-00-002. June 2001. http://www.epa.gov/safewater/ucmr/data/report_ucm1-2_ncod_summary.PDF

USEPA (2001b). United States Environmental Protection Agency. Occurrence of Unregulated Contaminants in Public Water Systems: An Initial Assessment. Office of Water. EPA 815-P-00-001 May 2001. http://www.epa.gov/safewater/ucmr/data/report_ucm1-2_no_uc.pdf

USEPA (2005a). Draft Risk Assessment of the Potential Human Health Effects Associated with Exposure to Perfluorooctanoic Acid and Its Salts. Office of Pollution Prevention and Toxics. January 4, 2005. http://www.epa.gov/ncea/raf/car2sab/guidelines_1986.pdf

USEPA (2005b). United States Environmental Protection Agency. Guidelines for Carcinogen Risk Assessment. Risk Assessment Forum. EPA/630/P-03/001F. March 2005. http://www.epa.gov/iriswebp/iris/cancer032505-final.pdf

USEPA (2006a). SAB Review of EPA's Draft Risk Assessment of Potential Human Health Effects Associated with PFOA and Its Salts. May 30, 2006. http://www.epa.gov/ncea/raf/car2sab/guidelines_1986.pdf

USEPA (2006b). United States Environmental Protection Agency. 2006 Edition of the Drinking Water Standards and Health Advisories. EPA 822-R-06-013. Office of Water. August, 2006. http://www.epa.gov/waterscience/criteria/drinking/dwstandards.pdf

USEPA (2007). United States Environmental Protection Agency. Integrated Risk Information System. 1,2,3-Trichloropropane. Last updated on 7/30/03. http://www.epa.gov/iriswebp/iris/subst/0200.htm

WHO (2003). World Health Organization. 1,2,3-Trichloropropane. (Concise international chemical assessment document; 56). http://www.inchem.org/documents/cicads/cicads/cicad56.htm